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REVIEW

Post-dural puncture headache

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ABSTRACT

Neuraxial analgesia and anesthesia are widely used in obstetric anesthesia. The most frequent complication after neuraxial blocks is post-dural puncture headache. It can occur after unintentional dural puncture during epidural procedures or after spinal anesthesia. Unintentional dural puncture occurs in 0.15-1.5% of labor epidural analgesia and 50-80% of these women develop post-dural puncture headache. The headache is typically orthostatic in nature and can be so incapacitating that the mother becomes bedbound and is no longer able to care for herself and her newborn child. A wide variety of prophylactic and therapeutic measures have been tried. So far, the therapeutic epidural blood patch is the only treatment for which there is enough evidence to recommend its routine use for severe cases of post-dural puncture headache. Larger multicenter trials are needed to back up alternative treatment strategies.

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Epidural analgesia offers the most effective form of pain relief during labour.[1] In high- and middle-income countries, the use of epidural labor analgesia has increased over the past decades. Rates vary greatly between countries due to availability and cultural habits. Accurate data on epidural rates are missing, but it is estimated that 30-80% of women in Europe and North America give birth with epidural analgesia. Post-dural puncture headache (PDPH) is a relatively frequent complication after neuroaxial blocks.[2] Anesthetists therefore need to be familiar with the clinical picture, differential diagnosis and treatment of PDPH. The aim of this review is to provide the reader with a comprehensive overview of the current literature.

History

The first account of successful neuroaxial anesthesia dates back to 1898, when Karl August Bier injected cocaine into the subarachnoid space of seven patients for lower extremity surgery. In 1901, Oskar Kreis, a Swiss obstetrician, performed the first neuroaxial block for pain relief in labor. He injected 10 µg of cocaine intrathecally at the level of L4-5 during the second stage of labor and observed pain relief within five to ten minutes. In these days, large spinal needles were used and PDPH was described to occur in 50% of all patients.[3] In 1909, Walter Stoeckel, a German obstetrician, published his experience with 141 cases of caudal epidural analgesia for labor pain. He injected procaine at the end of the first stage or the beginning of the second stage. With caudal injection, the risk of PDPH was much less.[4] The first epidural catheter technique for pain relief in labor was described in 1931 by Eugen Bogdan Aburel, a Romanian surgeon and obstetrician. Like Stoeckel, Aburel accessed the epidural space caudally. Only in the early 1960's did lumbar

epidural analgesia replace the caudal route, and lumber epidural catheters first came into use in the 1970's.[5]

Incidence

PDPH is a complication of dural puncture which occurs intentionally in spinal anesthesia or unintentionally as a complication of epidural anesthesia.

PDPH after epidural anesthesia

The incidence of unintentional dural puncture (UDP) during epidural anesthesia is described to be between 0.15% and 1.5%. In a systematic review published by Choi in 2002, the risk of unintentional dural puncture (UDP) during epidural insertion in parturients was described as 1.5%. Of these, around 50% developed a PDPH.[6] Sprigge analyzed data from almost 20,000 epidurals for labor analgesia performed over a time period of 23 years and found an incidence of 0.91% for recognized UDP with 88% developing a PDPH.[7] Tien reviewed data from over 40,000 epidurals performed for labor analgesia and identified UDP in 0.15%, PDPH developed in 63.1%.[8] The "serious complication repository project of the society for obstetric anesthesia and perinatology" collected data over a five-year period from 30 institutions in the USA and looked at the incidence of PDPH after all neuraxial anesthetics (epidural and spinal anesthesia). The authors reported an incidence for PDPH of 0.7%.[9] These numbers are helpful when consenting patients for epidural analgesia. The risk of developing a PDPH can be quoted as less than 1%.

PDPH after spinal anesthesia

The incidence of PDPH after spinal anesthesia varies greatly with the type of needle used. Choi *et al.* found an incidence of 1.5% to 11.2% in their meta-analysis.[6] Interestingly, they only found a significant difference for the type of needle, but not the diameter of the needle used. The risk to develop a PDPH was much higher when a cutting tip spinal needle was used, regardless of the diameter of the needle. If an atraumatic needle was used, the risk was much lower, again regardless of the needle diameter. These findings are in accordance with data published by Sprigge, who reported an incidence of PDPH of 3.5% if a 27- to 30-G Quinke spinal needle was used, but only 0.8% if a 24- to 26-G pencil point needle was used.[7] A systematic Cochrane review published in 2017 also came to the conclusion that the risk of developing a PDPH is much higher if a cutting tip needle is used instead of a pencil point needle, but did not find any significant difference when comparing different sizes of the same type of needle.[10] In our daily practice, we should therefore aim to use atraumatic needles for spinal anesthesia.

Risk factors

There are factors increasing the likelihood of an UDP occurring and there are risk factors increasing the chances of developing a PDPH once a dural puncture has occurred, either after a spinal anesthesia or as a complication during an epidural anesthesia.

The incidence of an UDP is inversely related to the operator experience, with less UDP occurring in experienced clinicians.[11-13] A greater degree of cervical dilatation at the time point of epidural insertion has also been shown to be associated with a higher rate of UDP.[14] The greater the degree of cervical dilatation, the more difficult it is for the laboring women to assume a favorable position and remain still for the time of epidural insertion.

Once UDP or intentional dural puncture (as in spinal anesthesia or lumbar puncture) have occurred, younger age, female gender, a history of prior PDPH and a history of chronic headache predispose the patient to the development of PDPH.[15-19] Pushing during the second stage of labor is also associated with a higher incidence of PDPH, more severe headache and increased need for an epidural blood patch (EBP) compared to women who deliver by cesarean section.[20-22] It is thought that by pushing, more cerebrospinal fluid is lost through the tear in the dura and therefore the pressure in the subarachnoid space drops more significantly, increasing the incidence and severity of the PDPH. Controversy exists regarding the effect of the Body Mass Index (BMI) on

the incidence of PDPH and the need for EBP. While Peralta describes a decreased incidence of PDPH after UDP and a decreased need for EBP in obese parturients,[23] several other studies did not find a difference.[24-26] The use of either air or saline for the loss of resistance technique did not make a difference on the incidence of UDP and/or PDPH according to a Cochrane systematic review.[27] Bevel orientation provides an opportunity to reduce the chance of developing a PDPH. During insertion and removal of a spinal needle, it is recommended that the bevel of any traumatic needle should be oriented parallel to the long axis of the spine in order to minimize injury of the parallel elastic fibers in the dura.[28] When performing an epidural, where no dural puncture is intended, we recommend to refrain from rotating the needle to minimize the risk of UDP or increasing the tear in case of an UDP. Strupp *et al.* found a reduced incidence of PDPH after lumbar puncture if the stylet of an atraumatic 21-G Sprotte needle was reintroduced before the needle was removed.[29] They hypothesized that the arachnoid mater could be attached to the tip of the needle after dural puncture and a small strand of it could be pulled out through the dural perforation when removing the needle and this could promote a prolonged CSF leak. The authors therefore recommend reintroducing the stylet before removing the needle to reduce the risk of an arachnoid strand being pulled out through the perforation site and therefore reduce the risk of PDPH. Further research is needed to see if this finding can be reproduced.

Clinical presentation

The Headache Classification Committee of the International Headache Society defines the PDPH in the third edition of the International Classification of Headache Disorders as an orthostatic headache caused by low cerebrospinal fluid pressure usually accompanied by neck pain, tinnitus, changes in hearing, photophobia and/or nausea. It occurs within five days of a lumbar puncture and is caused by cerebrospinal fluid leakage through the dural puncture. It remits spontaneously within two weeks, or after sealing of the leak with autologous epidural lumbar blood patch (Table I).[30] Sixty-six percent of the PDPHs start within the first 48 hours and about 90% within the first 72 hours after the dural puncture.[2, 3] The typical location of the headache is frontal and occipital with radiation into the neck and shoulder area. Loures *et al.* however found in a cohort of 142 parturients with PDPH that 5.6% presented with atypical non-postural headache. These women presented with stiffness and pain in the cervical, thoracic or lumbar vertebral area, visual disturbances and vertigo.[31]

TABLE I.—*Diagnostic criteria for post-dural puncture headache from the Headache Classification Committee of the International Headache Society.*[28]

Diagnostic criteria for PDPH
1. Orthostatic headache caused by low cerebrospinal fluid pressure and criteria (2) and (3) must be fulfilled
2. Dural puncture has been performed
3. Headache develops within five days of dural puncture
4. Accompanied symptoms (usually present, but not always)
• neck pain
• tinnitus
• changes in hearing
• photophobia
• nausea
5. Headache resolves either
• spontaneously within two weeks
• after sealing of the leak with autologous epidural lumbar blood patch

Differential diagnosis

Headaches during the postpartum period are very frequent. Goldszmidt *et al.* found an incidence of 39% during the first week postpartum in a prospective cohort study.[32] Thirty-eight percent were tension-type headaches, 27% were migrainous and 11% were musculoskeletal. Only 4.7% were PDPHs. Similar numbers were found by Stella *et al.* who analyzed retrospectively data from 95 women who presented with headaches >24 hours after delivery.[33] In this study, 39% of women presented with tension-type headache, 24% with preeclampsia/eclampsia, 16% with PDPH and 11% with migraine. In 10% of cases, a severe neurologic condition was the underlying cause for the headache such as pituitary hemorrhage/mass, cerebral venous thrombosis, cerebral vasculopathy or subarachnoid hemorrhage. Table II outlines the differential diagnosis of postpartum headache. Stella *et al.* proposed an algorithm for

TABLE II.—*Differential diagnosis of postpartum headache.*[30, 31]

Primary headache
• tension-type headache
• migraine
• musculoskeletal headache
• cluster headache
Secondary headache
• post-dural puncture headache
• preeclampsia/eclampsia
• cerebral venous thrombosis
• stroke (ischemic or hemorrhagic)
• ruptured aneurysm
• hypertensive encephalopathy
• pituitary apoplexy
• meningitis
• subarachnoid hemorrhage

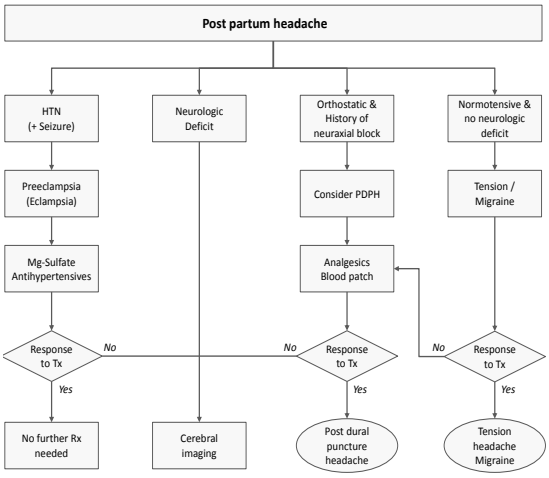

Figure 1.—Diagnostic algorithm for the management of severe postpartum headache (adapted from Stella *et al*.).[33]

the management of severe postpartum headache which lasts >24 hours after delivery (Figure 1). The key message is that women with headaches refractory to usual therapy and/or with focal neurologic deficits require cerebral imaging.

Complications

There are several case reports describing (rare) complications occurring after UDP. No data is available on the frequency of these complications. Subdural hematoma, diplopia as a consequence of cranial nerve palsy, cerebral venous thrombosis and postpartum depression have all been associated with UDP.[34-37] One case is described where a subdural hematoma after spinal anesthesia led to the death of the patient.[38] Both Webb *et al.* and Ranganathan *et al.* report a higher incidence of chronic headache and back pain in parturients with an UDP compared to controls (28% and 35% *vs*. 5% and 2% for chronic headache; 43% and 58% *vs*. 15% and 4% for chronic back pain).[39, 40] Interestingly, Webb *et al.* found that the incidence of chronic headache was lower when PDPH was treated by epidural blood patch than by conservative measures and treatment with an epidural blood patch did not increase the risk for chronic back pain compared to women with UDP who were not treated with an EBP.

Pathophysiology

Cerebrospinal fluid (CSF) is produced mainly in the choroid plexus of the ventricles of the brain and reabsorbed by arachnoid granulations (little protrusions of the arachnoid mater) into the blood stream. The daily production of CSF is about 500 mL and the average CSF volume in the adult is 150 mL of which half is in the cranial cavity and half in the spinal cavity.[3] If there is a perforation in the lumbar dura large enough that the CSF leak is greater than the CSF production, the CSF pressure will drop. Orthostatic headache is estimated to occur if more than 10% of the total CSF volume is lost.[2] There are two postulated mechanism through which CSF hypotension causes headache. One theory makes the downward pull on pain sensitive structures in the brain responsible for the development of the orthostatic headache. If the patient assumes an upright position, CSF is shifted from the cranial cavity to the vertebral canal. Due to the reduced volume of CSF, the brain sags into the foramen magnum pulling on the meninges, vessels and nerves. The sagging of the brain can also lead to a compression of the cranial nerves and explains why some patients experience cranial nerve palsies. The second postulated theory uses the Kellie-Monroe doctrine as an explanation for the PDPH. If CSF is lost, cerebral vasodilation must occur in order to keep the total intracranial volume constant. The cerebral vasodilatation is made responsible for the headache. For both theories, there is radiologic evidence in cranial MRI scans.[2, 3]

Prevention

Several interventions have been proposed to prevent the occurrence of PDPH after intentional or unintentional dural puncture, some of them proved beneficial, whereas others did not show any benefit or even proved to increase the risk of developing PDPH. In the following paragraph, we will explore the available evidence for the most common preventive strategies discussed in the literature.

Bed rest

A Cochrane systematic review did not find any benefit in preventive bed rest after dural puncture. Bed rest probably increased the risk of developing PDPH compared to immediate mobilisation.[41]

Fluid supplementation

The same Cochrane systematic review analyzed the effect of fluid supplementation on the prevention of PDPH. There was low quality evidence for an absence of benefits on the incidence of severe PDPH.[41]

Neuraxial opioids

In a small prospective, randomized, double blind study Al-Metwalli was able to show a significant reduction in the occurrence of PDPH and the need for an EBP after UDP in parturients, if they received two epidural injections of 3mg morphine each, 24 hours apart.[42] In contrast, spinal morphine and spinal fentanyl did not show any preventive benefit. Epidural morphine unfortunately increased the number of women affected by nausea and vomiting.[43]

Intravenous cosyntropin

Cosyntropin is an analog of ACTH and is normally used to diagnose an insufficiency of the adrenal glands (Synacthen test). One prospective randomized, controlled trial found a reduction in the incidence of PDPH and need for EBP after UDP in parturients who received 1mg of cosynthropin intravenously compared to placebo.[44]

Intravenous dexamethasone

Two studies assessed the preventive benefit of intravenous dexamethasone. One study did not find any benefit when dexamethasone was given to patients who underwent spinal anesthesia for lower extremity orthopedic surgery.[45] The other study found an increased incidence of PDPH after intravenous dexamethasone given to women who underwent spinal anesthesia for cesarean section.[46]

Oral caffeine

A Cochrane systematic review found no reduction in the occurrence and severity of PDPH if women were prescribed prophylactic caffeine after dural puncture. As an unwanted side effect, it increased insomnia.[43]

Prophylactic epidural blood patch

If an UDP occurred during epidural anesthesia, there are two possibilities to proceed. Either the catheter is threaded through the perforation into the intrathecal space, or the Tuohy needle is withdrawn, and the epidural catheter is sited at a different intervertebral space. If the second possibility is chosen, a prophylactic epidural blood patch can be performed by injecting blood through the epidural catheter after resolution of the epidural block and before removal of the epidural catheter. There is much controversy if this technique is effective in reducing the incidence of PDPH. A systematic review published by Apfel in 2010 included nine studies, five non-RCTs, and four RCTs. Pooled results from the five non-RCTs demonstrated a significant risk reduction in the incidence of PDPH (RR 0.48 [0.23-0.99]), the four RCTs however did not show a significant risk reduction (RR 0.32 [0.10-1.03]). Study protocols and results were very heterogenous.[47] Since then, a randomized controlled, but not blinded, trial by Stein included over 100 parturients with UDP and assigned them to either prophylactic EBP or conservative treatment. In the prophylactic EBP group 18.3% developed a PDPH, in the group with conservative treatment 79.6% developed a PDPH. 73.4% of the women in the group with conservative treatment required a therapeutic epidural blood patch, and of these 11.1% required a second EBP. In the prophylactic EBP group, 10% required a therapeutic EBP after the initial prophylactic blood patch.[48] In summary it can be said that the studies sup-

porting the use of prophylactic EBP were either non controlled or had methodological limitations. Therefore, there is currently insufficient evidence to recommend the use of prophylactic EBPs.

Intrathecal catheter insertion

As mentioned before, it is a possibility to thread an intrathecal catheter through the dural rent after UDP. The risk of a second UDP if re-siting the epidural at a different level is quoted at 4-9%.[49-51] Inserting an intrathecal catheter omits this risk. It can be used for labor analgesia and toped up for a Cesarean section. A retrospective review of 761 intrathecal catheters used in obstetric patients did not find any serious complications.[26] Jagannathan retrospectively reviewed 235 parturients where an UDP had occurred. One hundred and seventy-three women received an intrathecal catheter and in 63 women, the epidural was re-sited. The choice of neuraxial technique (spinal catheter *versus* epidural re-site) did not have any influence of the course of labor. Intrathecal catheters did neither prolong the second stage of labor nor led to a higher rate of cesarean sections.[22] Data on failed labor analgesia with an intrathecal catheter are conflicting. Cohn reports a rate of 6.1% of failed labor analgesia after intrathecal catheter placement following UDP. No control group of re-sited epidural catheters was available in this study.[26] Jagannathan *et al.* found a much higher rate of failed labor analgesia after intrathecal catheter placement compared to epidural re-siting (14% *vs.* 2%).[22] Tien *et al.* found a failure rate of 22% for intrathecal catheters and 13% for re-sited epidurals. Their results did not reach statistical significance due to a small number of women included.[8] Verstraete *et al.* reported that there was no case of failed labor analgesia in their cohort of 89 obstetric patients who received an intrathecal catheter following UDP.[52] Equally conflicting are the data regarding the potential benefit of reducing the incidence of PDPH and need for EBP with an intrathecal catheter. Most studies are retrospective and analyze only small number of patients. Verstraete *et al.* report that placing an intrathecal catheter and leaving it for at least 24 hours postpartum reduces the rate of PDPH significantly (61% *vs.* 48%, P=0.04) and results in a trend towards reduction of the need for an EBP (54% *vs.* 36%, P=0.06).[52] The idea behind inserting an intrathecal catheter and leaving it for 24 hours is that the catheter seals the rent created by the UDP and prevents CSF from leaking. Additionally, it causes a local irritation of the perforation site which might expedite healing. Other studies report that insertion of an intrathecal catheter does not result in a reduction in the incidence of PDPH, but reduces the need for a therapeutic EBP.[3, 49, 53, 54] A third group of studies does not report a significant difference for either PDPH or EBP.[8, 50] The only prospective study comparing intrathecal catheters with epidural re-site, did not find a significant difference[51] and a systematic review published by Apfel *et al.* in 2010 came to the same conclusion.[47] A systematic review from 2013, found a significant reduction in the need for an EBP, but no significant reduction in the occurrence of PDPH. However, after excluding a study by Ayad *et al.* which reported a much higher beneficial effect of an intrathecal catheter than all the other published studies, the beneficial effect of the intrathecal catheter disappeared.[55] The biggest danger of intrathecal catheters is accidental administration of an epidural dose and resulting high block.[26, 50] However, high blocks have also been reported after epidural re-site following UDP,[50] most likely caused by a diffusion of the epidural drug through the dural rent into the intrathecal space.

Therapeutic management

There is almost no limitation to what has been tried in the treatment of PDPH. Unfortunately, neither conservative nor pharmacological measures have been promising. In order to treat the PDPH effectively, the lost CSF must be replaced, the rent in the dura must be sealed and the cerebral vasodilatation controlled.

Simple measures

Bed rest, rehydration, paracetamol, non-steroidal anti-inflammatory drugs, opioids and antiemetics can help relieve symptoms, but are only supportive and not curative measures. Abdominal binders have been tried to alleviate the pain by increasing the intraabdominal pressure and

therefore also the pressure in the epidural space. They are however not practical in the postpartum period.[3, 28, 56]

Caffeine

Caffeine causes a vasoconstriction of dilated cerebral vessels and increases CSF production. It is therefore used in the treatment of PDPH. Intravenous and oral formulations at a dose of 300-500 mg daily have been tried. Caffeine has an oral bioavailability of almost 100%, therefore one route is not superior to the other. A systematic Cochrane review and a review by Katz *et al.* came to the conclusion that caffeine can reduce the pain score temporarily and therefore might reduce the need for further treatment.[56, 57]

Other pharmacological treatment options

For theophylline, aminophylline, gabapentin and pregabalin a reduction in pain scores has been reported. The data for cosyntropin is highly conflicting, most likely it has more value in preventing PDPH than in its treatment. There is no evidence for the therapeutic benefit of sumatriptan.[56, 57]

The epidural blood patch is the most effective treatment option of severe PDPH. Success rates of 70-98% after a first epidural blood patch have been reported with similar results if the procedure is repeated.[3, 28, 58] Therefore a special section is dedicated to the EBP.

Epidural blood patch

The first epidural blood patch was performed in the 1960's after the observation that bloody taps were associated with a reduced PDPH rate.[3] The mechanism by which the EBP relieves the symptoms of PDPH is most likely multifactorial. Beards performed MRI scans of patients between 30 minutes and 18 hours after EBP.[59] During the first three hours, there is a significant mass effect from the injected blood, compressing the thecal sack and spinal nerves. The mass effect was greatest at the injection site, but the blood spread cephalic and caudally as well as to the anterior epidural space. The compression during the first hours explains the almost instant relief most patients experience after epidural blood injection. CSF is pushed into the cranium, increasing the intracranial pressure and causing a reflex vasoconstriction of the cerebral vessels. The blood clot seals the rent in the dura, preventing further leaking of CSF. The CSF volume is restored at a rate of around 20 mL/h. The resolution of the symptoms at later time points, once the mass effect of the injected blood has worn off (visible in the MRI images from seven hours onwards) can be explained by the restoration of the CSF volume.

The optimal timing of the EBP has been subject to much discussion. There are studies supporting that delaying the blood patch for 48[60] or 96 hours[58] after UDP increases the success rate. The theory behind this practice is that epidural saline from the loss of resistance technique and CSF from the leak dilute the blood injected into the epidural space and reduce the effectiveness of the EBP. However, one can argue that patients with bigger dural defects and therefore more CSF leakage, will present earlier with severe symptoms of PDPH and require an EBP earlier. In these women, the success rate of the EBP might be reduced simply due to the size of the dural lesion. Armstrong *et al.* studied the effect of serial *in-vitro* hemodilution with CSF and crystalloid on thrombo-elastographic blood coagulation parameters. They found that the presence of both CSF and crystalloids had significant procoagulant effects (decrease in r-time [time until initial fibrin formation], decrease in k-time [time to achieve a certain level of clot strength] and increase in α-angle [speed at which fibrin builds up]), but destabilized the clot strength (decrease in the maximal amplitude). The effect of CSF on the clot was more pronounced than the effect of cristalloids.[61] A PDPH often leaves the new mother incapable of caring for herself and her baby. It therefore seems unjustified to withhold EBP for 48 hours or longer, only because there is a higher chance that the procedure needs to be repeated.

Peach performed a much cited randomized blinded trial to determine the optimal blood volume for epidural blood patches in obstetrics.[62] Women received either 15, 20, or 30 mL of autologous blood. In the 15-, 20-, and 30-mL groups the incidence of permanent or partial relief was

61%, 73%, and 67%, respectively. Complete relief was achieved in 10%, 32% and 26%. These findings are in accordance with another study by Booth where investigators aimed to inject 30 mL of blood but would stop injection if the patient experienced pain. The mean volume injected was 20.5 mL and the investigators did not find an increase in success rate with increasing blood volumes.[63] The optimal volume of blood still remains to be determined and is hardly the same for every patient. Current findings however support a practice of aiming to inject 20 mL but stopping earlier if the patient complains of back pain.

With this practice, complete and partial relief of symptoms after EBP can be expected to be 32% and 73% respectively.[62] About one-third of parturients require a second EBP.[64] Most agree that cranial magnetic resonance imaging should exclude intracerebral pathologies (such as subdural hematoma) before a third EBP is performed.

EBP is considered a safe procedure. No exact numbers on the incidence of adverse events are available. There are however a number of case reports describing complications after EBP. Inadvertent subdural blood injection leading to severe lumbar back pain and radicular symptoms have been described. Symptoms resolved spontaneously in all cases.[65-67] Initial exacerbation of PDPH symptoms after EBP which responded to treatment with non-steroidal anti-inflammatory drugs has also been described.[68] There is an arbitrary fear among anesthetists that the blood injected into the epidural space during an EBP could act as a fertile soil for bacterial growth. Interestingly, there are no cases of epidural abscess after EBP described in the literature. As long as a sterile technique is applied, the risk of infection appears to be negligible.

Alternative treatment options

A number of alternative treatment options have been described. For none of them, enough evidence exists to support their routine use. They should therefore only be used for special indications.

Epidural saline

Several regimes have been proposed ranging from a single injection of 30 mL after the development of PDPH to a continuous prophylactic infusion of 1-1.5 liters of epidural saline over 24 hours. Transient benefits have been observed, most likely due to epidural mass effect. Because saline does not stay in the epidural space, the effect disappears once the saline has diffused away. Administration of large volumes of epidural saline is not without risk and can cause intraocular hemorrhages through a rise in intracranial pressure.[3, 56]

Epidural hydroxyethyl starch

Like for epidural saline, a transient benefit has been observed. There is no evidence for the hypothesis that hydroxyethyl starch remains any longer in the epidural space than epidural saline.[3, 56]

Acupuncture

Acupuncture has proven beneficial in a number of small case series.[69-71] Large studies are missing.

Greater occipital nerve blocks

The greater occipital nerve is a sensory nerve originating from C2-C3. This nerve can be blocked lateral to the nuchal midline and medial to the occipital artery. It blocks sensations from the skin, muscles and vasculature over the posterior aspect of the head. Greater occipital nerve blocks have been used successfully in the treatment of migraine and cluster headaches.[56] Small studies have shown a beneficial effect in the treatment of PDPH.[72, 73]

Transnasal sphenopalatine ganglion block

The sphenopalatine ganglion is located in the pterygopalatine fossa in the posterior nasal pharynx and contains sympathetic, parasympathetic and sensory nerve fibers. It can be blocked by inserting cotton-tipped applicators soaked in local anesthetic through the nasal cavity. Successful treatment of PDPH has been described.[74, 75]

Neostigmine and atropine

A recently published randomized, controlled, double-blinded trial studied the benefit of intravenous neostigmine and atropine in the treatment of PDPH after spinal anesthesia for elective cesarean section. Women in the study group received 20 μg/kg neostigmine and 10 μg/kg

atropine IV every eight hours, women in the control group received intravenous saline only. Treatment was stopped once the pain score was ≤3. Women who still had a pain score ≥5 after 72 hours received an EBP. Pain scores at six, 12, 24, 36, 48, and 72 hours after intervention were significantly lower in the study group and none of the women in the study group required an EBP, whereas 16% of the women in the control group required an epidural blood patch. All women in the neostigmin/atropine group achieved a pain score of ≤3 after two doses. There were however significant side effects to the treatment with neostigmine/atropine. Twenty percent of women experienced abdominal cramps, 15% muscle twitches, and 12% urinary bladder hyperactivity. None of these side effects were observed in the control group. Because of the high number of women with abdominal cramps and urinary bladder hyperactivity and the fact that women cannot breastfeed for 24 hours after neostigmine, we question the benefit of this treatment for obstetric patients. Further studies are needed to see if neostigmine/atropine is beneficial to patients with PDPH outside the obstetric population.[76]

Conclusions

PDPH is one of the most common complications of neuraxial anesthesia, but it is still rare. It is therefore difficult to conduct well designed large randomized controlled trials. Existing data comes from small, often retrospective, studies or case series. Current practice is backed up more by expert opinion than strong evidence. Until new data from large multicenter studies is available, it is advisable to adhere to existing protocols. After UDP, insertion of an intrathecal catheter should be at least considered. Even if its beneficial role in preventing PDPH and need for EBP is controversial, inserting an intrathecal catheter reduces the risk of a second UDP. At the time being, there is no evidence for any prophylactic treatment. Once PDPH has occurred, relieving symptoms with paracetamol, non-steroidal anti-inflammatory drugs, opioids and caffeine can be tried. If the PDPH is compromising a woman's ability to look after herself and her baby, EBP should be offered early if no contraindications exist. If the patient refuses an EBP or if there are contraindications, alternative treatment options should be considered.

Key messages

- Unintentional dural puncture occurs in 0.15-1.5% of labor epidural analgesia and 50-80% of these women develop PDPH.
- Once an unintentional dural puncture has occurred, younger age, female gender, a history of prior chronic headache and pushing during the second stage of labor (compared to delivery by cesarean section) predispose patients to the development of a PDPH.
- Almost 40% of women experience headache pain during the first postpartum week, but in less than 5% of postpartum women with headaches, spinal hypotension is the underlying cause. Clinicians involved in peripartum care must be familiar with the differential diagnosis of PDPHs.
- To date, none of the prophylactic measures tried to reduce the incidence of PDPH after intentional or unintentional dural puncture, have showed significant benefit and can be recommended for daily clinical use.
- The epidural blood patch still remains the gold standard for treatment of PDPHs with complete relief of symptoms in 32% and partial relief in 73%.

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